## Inclusion Bodies of the Erythrocyte

SIR—With reference to your annotation (Aug. 28, p. 431) entitled "Inclusion Bodies of the Erythrocyte," we should be obliged if you would allow us to refute an opinion you attribute to us. Referring to our publication on this subject you state, "They are convinced that the siderotic granules of Grüneberg are different from the Pappenheimer bodies. . . ." This is, indeed, a complete misrepresentation of our views, for on page 255 of the paper referred to[1] we stated, "In view of these considerations it would appear that the siderotic granules described by Grüneberg (1941a and b, 1942) and by Doniach *et al.* (1943) are probably fundamentally similar to the Pappenheimer bodies."

On the other hand, we wish to emphasize our belief that these inclusion bodies, which stain with Romanowsky stains and give a direct positive reaction with potassium ferrocyanide and hydrochloric acid, are fundamentally dissimilar from the siderotic granules described by Case.[2 3 4] The arguments supporting this belief were discussed in our paper, but it may be mentioned here that Case's granules differ in respect of occurrence, morphology, and staining reactions from the erythrocytic inclusions described by Grüneberg in curly-tailed mice, and by Pappenheimer *et al.* and by ourselves[1] in man.

Your annotation also refers to our having collected some evidence that the granules of basophilic stippling due to lead poisoning are similar to Pappenheimer bodies. It may be of interest to note that we have extended our studies in this field and have confirmed that the basophilic granules of lead poisoning present many similarities to the Pappenheimer bodies as regards development, staining reactions in the bone marrow and in the peripheral blood, and the effect of splenectomy. This work has recently been submitted for publication.—We are, etc.,

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Glasgow. L. J. DAVIS.

REFERENCES

1 *Glasg. med. J.*, 1947, **28**, 237.
2 *Nature*, 1943, **152**, 599.
3 *J. Path. Bact.*, 1945, **57**, 271.
4 *Proc. roy. Soc. B.*, 1946, **133**, 235.
5 *J. Genet.*, 1942, **44**, 246.
6 *Quart. J. Med.*, 1945, n.s., **14**, 75.

## Production of Rh Agglutinins

SIR,—I must apologize to Dr. R. J. Drummond (Aug. 28, p. 443) and Drs. Sheila T. Callender and Z. V. Paykoç (Jan. 26, 1946, p. 119) for giving the impression that they suggested that "patients suffering from certain diseases were more apt to produce antibodies." This idea was communicated to me in conversation, but I have quite forgotten with whom, or who originated the idea—it may even have been myself. I still think it is a hypothesis worth considering, and if anyone who has suitable patients available is able to test the idea the results, whether positive or negative, would be interesting.

I must apologize for a *lapsus ratiocinationis* pointed out by Professor Cappell—the product of the mating Rr × rr is equal numbers of Rr and rr—not 3 rr to 1 Rr as we wrote. This error does not affect our argument or conclusions.—I am, etc.,

London, N.W.10. GEORGE DISCOMBE.

## Acute Porphyria

SIR,—I read with great interest Dr. Ernest Petrie's article (May 15, p. 926) on "A Case of Acute Porphyria." The article reminded me of a similar case published by me in 1917 (*Z. klin. Med.*, **84**, 3) which showed the typical symptoms of chronic porphyria, including a positive benzaldehyde (Ehrlich's) test. Owing to some unusual features I should like to give a short summary of the case.

A 24-year-old girl was admitted to hospital suffering from frequent attacks of severe abdominal colic; negative family history. Case history: temporary amenorrhoea and irregular menses; had been suffering from these attacks for last 4 years, thought by some doctors to be due to peptic ulcer, by others to be of nervous origin. After the attacks she passed an acid cherry-red urine, darkening on standing, and becoming brown-red on longer standing. Trace of albumin, hyaline and granulated casts in the deposit. On addition of alkali the colour turned yellow, but on acidification the red colour returned. Negative guaiac and distinctly positive Ehrlich's benzaldehyde tests. Pigment not soluble in ether. Prof. Salkowski, who examined the urine, reported: pigment soluble in amyl alcohol, slightly soluble in acetic ether, positive Ehrlich test but negative urobilin test; spectroscopically the acid urine gave a diffuse absorption band in the region of the urobilin band. Salkowski came to the conclusion that the abnormal pigment was "probably so-called 'skatol-red,' which, however, was not sufficiently well characterized." Hot baths with subsequent diaphoresis seemed to relieve the pain. Patient died 1½ months later in a coma which occurred suddenly with epileptiform convulsions in all extremities, jerking of the head to the left, and horizontal nystagmus.

The results of post-mortem examination were: atrophy of musculature, dehydration, slight oedema of pia, hypoplastic aorta, small heart with moderately hypertrophied left ventricle. Pathologist's report: cortex of kidneys much reduced; in periphery tubuli very much reduced, fibrosis and small-cell infiltration; slight fatty degeneration in tubuli but more so in Henle's loops; a few glomeruli showing amyloid degeneration (these findings did not, in his opinion, justify the diagnosis "death from uraemia"); fatty degeneration in most lobuli of liver, slight proliferation and small cell infiltration of peripheral fibrous tissue.

In the light of present knowledge of porphyrins the described data—red urine pigment not soluble in ether but soluble in amyl alcohol and slightly soluble in acetic ether, negative urobilin and positive benzaldehyde tests, absorption band in the region of urobilin—seem, no doubt, to justify the diagnosis of coproporphyrin porphyria.—I am, etc.,

London, N.W.11. M. G. GOOD.

## Sturge-Kalischer-Weber Syndrome

SIR,—Dr. C. Worster-Drought (Aug. 28, p. 414) gives a valuable summary of the literature of this syndrome and describes a case of naevoid amentia with bilateral calcification of cerebral vessels.

A boy, aged 16½ years, was admitted to Cell Barnes Colony in 1939 and died of pneumonia in 1940. He was a quadriplegic epileptic idiot, with but one cutaneous naevus above the nose and practically central. Radiographic examination showed evidence of much naevus on the left side and some to the right of the centre line. The specimen itself shows more than the radiograph suggests, the condition being most marked in the left temporo-sphenoidal and occipital regions, but also present in the right frontal and parietal areas. This is the only true case seen at the Colony out of 1,300 admissions.

Another has the typical cutaneous naevus, widely distributed, right more than left, and is of imbecile grade. There is some evidence of paresis, particularly on the left, but no fits and no *x*-ray evidence of meningeal naevus. She is regarded as a probable but not proven example of naevoid amentia.—I am, etc.,

St. Albans. NOEL H. M. BURKE.

## Dental Caries in Children

SIR,—Lady Mellanby and Dr. Helen Mellanby (Aug. 28, p. 409) claim to have shown that better-formed teeth are less liable to decay. If they mean that such teeth are less liable *because* of their better structure their data seem to disprove the claim. They suggest that the lessened caries incidence in 1947 compared with that in 1943 is due to the better calcifying properties of the diet, and they also state that the actual factors initiating caries remain obscure.

To come to such conclusions they must ignore not only most of the work of other students of the subject but also some of their own findings. Although the data are not statistically analysed, it is apparent from Table IV that there is a very close correlation between hypoplasia and high caries incidence in 1943, but in 1945 this correlation is slightly reduced, and in 1947 it is very much weaker and possibly insignificant. This is because the caries incidence on the better-formed teeth has actually *increased* by 1947. The better the structure the greater is the increase, and the worse the hypoplasia the greater is the decrease in caries. Both increases and decreases between 1943 and 1947 appear to be significant for each grade of tooth structure. In other words, the decrease in hypoplasia incidence by 1947 has been most marked in the carious teeth and least in the non-carious teeth. It follows that improved structure cannot account for most of the decrease in caries, but only a statistical analysis could show how much, if any, it can account for. Certainly some other factors are responsible for much of the improvement, and these factors, which seem to